A PROSPECTIVE RANDOMISED STUDY ABOUT ATTENUATION OF HAEMODYNAMIC CHANGES DURING LARYNGOSCOPY AND ENDOTRACHEAL INTUBATION WITH INTRAVENOUS LIDOCAINE VERSUS INTRAVENOUS DEXMEDETOMIDINE

Dissertation Submitted to

THE TAMIL NADU DR. MGR. MEDICAL UNIVERSITY,
CHENNAI–600032. TAMILNADU.

In partial fulfillment of the regulations
For the award of the degree of

M.D. DEGREE BRANCH-X
ANAESTHESIOLOGY

April 2017 Examinations

GOVERNMENT MOHAN KUMARA MANGALAM
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GOVERNMENT MOHAN KUMARAMANGALAM MEDICAL COLLEGE & HOSPITAL

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Signature of the Candidate

Place:

Dr. Ramesh. S

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Dr. Ramesh. S

PG. Ramesh – Dessertation

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Dated: 29 .06.2015.

Ethical Committee Meeting held on 18.06.2015 at 10.00 A.M in the Seminar Hall, IInd Floor, Medicine Block, Govt. Mohan Kumaramangalam Medical College Hospital, Salem - 01.

The following Members were attended the Meeting.

MEMBERS:

1. Dr. V. Dhandapani, MD., Deputy Chairman, External Social Scientist, ECIRB.
2. Dr. S. Mohamed Musthafa, MD., Vice Principal, Govt. Mohan Kumaramangalam Medical College, Salem.
3. Mr. S. Shanmugam, B.Sc., BL, Advocate, External Legal Expert.
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Sl. No.	Name of the Presenter with Address	Title	Name of the Guide and Address	Whether it is Approved or not.
1.	Dr. S. Ramesh, II Year, Post Graduate Student of MD (Anaesthesiology), GMKMC, Salem-30.	"A prospective randomised study about attenuation of haemodynamic changes during laryngoscopy and endotracheal intubation with intravenous lidocaine versus intravenous dexmedetomidine".	Dr. R. Nagarajan, MD., Professor of Anaesthesiology, GMKMC, Salem-30.	Approved

The Ethical Committee examined the studies in detail and is pleased to accord Ethical Committee approval for the above Post Graduate student of this College to carry out the studies with the following conditions.

1. He should carry out the work without detrimental to regular activities as well as without extra expenditure to the institution to Government.
2. He should inform the institution Ethical Committee in case of any change of study procedure site and investigation or guide.
3. He should not deviate from the area of the work for which applied for Ethical clearance. He should inform the IEC immediately, in case of any adverse events or serious adverse reactions.
4. He should abide to the rules and regulations of the Institution.
5. He should complete the work within the specific period and if any extension of time is required he should apply for permission again and do the work.
6. He should submit the summary of the work to the Ethical Committee on completion of the work.
7. He should not claim any funds from the institution while doing the work or on completion.
8. He should understand that the members of IEC have the right to monitor the work with prior intimation.

for DEAN 29/06/2015

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DECLARATION BY THE CANDIDATE

I hereby declare that this dissertation titled "**A PROSPECTIVE RANDOMISED STUDY ABOUT ATTENUATION OF HAEMODYNAMIC CHANGES DURING LARYNGOSCOPY AND ENDOTRACHEAL INTUBATION WITH INTRAVENOUS LIDOCAINE VERSUS INTRAVENOUS DEXMEDETOMIDINE**" is a bonafide and genuine research work carried out by me under the guidance of **Dr. R. NAGRAJAN M.D., Professor**, Department of Anesthesiology, Government Mohan Kumaramangalam Medical College Hospital, Salem, Tamil Nadu, India.

Date: 30.9.16
Place: SALEM

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INTRODUCTION

Direct laryngoscopy and endotracheal intubation forms the vital step for providing general anaesthesia. Laryngoscopy and intubation is a adverse stimulus, which can cause unaccepted response in the respiratory, cardiovascular systems [(1)]. Tachycardia and hypertension accompanyng direct laryngoscopy and intubation in the light plane of anesthesia has been reported since 1950 [(2)]. The magnitude of cardiovascular response is directly related to the force and duration of laryngoscopy [(3)]. Tachycardia and hypertension in retort to laryngo-tracheal stimulation are due to reflex sympathetic discharge, which in sequence causes increased plasma norepinephrine concentration [(4)].

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ABSTRACT

INTRODUCTION

Haemodynamic changes like hypertension & tachycardia in response to the stress to instrumentation of larynx and trachea namely; direct laryngoscopy & endotracheal intubation have been reported. This is mainly due to reflex sympathetic discharge in responses to laryngotracheal stimulation. This is usually transient and variable, and are well tolerated by healthy individuals. However, these changes may be fatal in patients with hypertension, coronary artery diseases and intracranial hypertension.

AIMS AND OBJECTIVES

"to compare the attenuation of haemodynamic changes during laryngoscopy and endotracheal intubation with intravenous lidocaine versus intravenous dexmedetomidine".

MATERIALS AND METHODS

study – randomized, double blind.

ethical committee approval - obtained from my institute,

written informed consent - obtained from all the patients.

SOURCE OF DATA:

Sixty one patients of both sexes admitted for elective surgeries under general anaesthesia in various surgical disciplines of GMKMCH, SALEM.

OBSERVATION:

From our study, we observed that lignocaine attenuated but did not fully abolish the pressure response to laryngoscopy and intubation. Also we adequately establish that dexmedetomidine 1μg/kg was comparatively superior in attenuation of the haemodynamic changes during direct laryngoscopy.

CONCLUSION

We conclude that dexmedetomidine in the dosage of 1 μg/kg over ten minutes before intubation efficiently attenuating the haemodynamic changes to laryngoscopy and endotracheal intubation. Lignocaine in the dosage of 1.5 mg/kg given 3 min before laryngoscopy and intubation was not fully effective in reducing the increase in heart rate and blood pressure.

Hence Dexmedetomidine may beneficial for cardiac patients where the haemodynamic response to laryngoscopy and intubation is highly detrimental.

KEYWORDS: Catecholamine release; Dexmedetomidine; Hemodynamic response; Lignocaine; Pressor response

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0 DBP	Post Intubation Diastolic Blood Pressure at 0 min
0 HR	Post Intubation Heart Rate at 0 min
0 MBP	Post Intubation Mean Blood Pressure at 0 min
0 SBP	Post Intubation Systolic Blood Pressure at 0 min
1 DBP	Post Intubation Diastolic Blood Pressure at 1 min
1 HR	Post Intubation Heart Rate at 1 min
1 MBP	Post Intubation Mean Blood Pressure at 1 min
1 SBP	Post Intubation Systolic Blood Pressure at 1 min
3 DBP	Post Intubation Diastolic Blood Pressure at 3 min
3 HR	Post Intubation Heart Rate at 3 min
3 MBP	Post Intubation Mean Blood Pressure at 3 min
3 SBP	Post Intubation Systolic Blood Pressure at 3 min
5 DBP	Post Intubation Diastolic Blood Pressure at 5 min
5 HR	Post Intubation Heart Rate at 5 min
5 MBP	Post Intubation Mean Blood Pressure at 5 min
5 SBP	Post Intubation Systolic Blood Pressure at 5 min
ASA PS	American Society of Anaesthesiologist Physical Status
BDBP	Basal Diastolic Blood Pressure
BHR	Basal Heart Rate
BMBP	Basal Mean Blood Pressure
BMI	Body Mass Index
BSBP	Basal Systolic Blood Pressure
CAD	Coronary Artery Disease
CRF	Chronic Renal Failure
HT	Height
PLDBP	Pre Laryngoscopy Diastolic Blood Pressure
PLHR	Pre Laryngoscopy Heart Rate
PLMBP	Pre Laryngoscopy Mean Blood Pressure
PLSBP	Pre Laryngoscopy Systolic Blood Pressure
WT	Weight

INTRODUCTION

Direct laryngoscopy and endotracheal intubation forms the vital steps for providing general anaesthesia. Laryngoscopy and intubation is a adverse stimulus, which can cause unaccepted response in the respiratory, cardiovascular systems.[1] Tachycardia and hypertension accompanying direct laryngoscopy and intubation in the light plane of anesthesia has been reported since 1950.[2] The magnitude of cardiovascular response is directly related to the force and duration of laryngoscopy.[3] Tachycardia and hypertension in retort to laryngo-tracheal stimulation are due to reflex sympathetic discharge, which in sequence causes increased plasma norepinephrine concentration.[4]

The sympathoadrenal response occurs after laryngoscopy and intubation peaks at 1.2 minutes and returns to baseline within 5 to 10 minutes. Though these sympathoadrenal responses are probably of little significance in healthy individuals, it is hazardous to those with hypertension, coronary artery heart disease, intracranial pathology and hyperactive airways. In such cases, these responses need to be suppressed.

Tachycardia, hypertension and dysrhythmias all occur during laryngoscopy and intubation.[5] The consequent increase in Rate Pressure

Product may result in a myocardial oxygen demand exceeding the myocardial oxygen supply resulting in myocardial ischemia. This response are sympathetically mediated. Prof King et al[6] documented myocardial ischaemic changes due to reflex sympathoadrenal response immediately following laryngoscopy and intubation with a mean increase in systemic pressure of 40mm Hg even in normotensive patients.

An increase in heart rate is more likely to produce signs of myocardial ischemia on ECG than hypertension. Indeed, in anaesthetized patients, the incidence of myocardial ischaemia on the ECG increases in patients who experience a heart rate greater than 110 beats per minute (Ischaemic Threshold). A frequent recommendation is to keep up the heart rate and blood pressure within 20% of the normal awake value for that patient.

Many attempts were made to attenuate the hemodynamic response to direct laryngoscopy and intubation. They were:

1. Intubating in a deep plane of anaesthesia[7]
2. Use of ganglionic blockers[8]
3. Use of topical anaesthesia[9,10]
4. Use of arterial dilators – hydrallazine, SNP[11]

5. Use of venodilators – NTG[12]
6. Use of magnesium sulphate
7. Use of beta blockers – Esmolol[13]
8. Use of Ca channel blockers[14]
9. Use of opioids – Fentanyl, Morphine, Pethidine[15,16,17]
10. Use of intravenous local anaesthetics

All the methods detailed above have their own advantages and disadvantages. Lignocaine is one of the most widely used drug in the group of local anesthetics, which is an aminoethylamide and prototype of amide local anesthetic group. In 1961, Bromage found that intravenous (IV) lignocaine can blunted pressor response during laryngoscopy and intubation.[18] Intravenous form of lignocaine is easily available and most commonly used drug to attenuate laryngoscope and intubation. Dexmedetomidine, a α_2 adrenergic agonist, has anesthetic sparing, analgesic, sedative, anxiolytic and sympatholytic effects. It decreases CNS sympathetic discharge in a dose-dependent manner. It has analgesic effects which is best defined as opioid sparing. In view of the fact that dexmedetomidine has shown minimal side-effects and its efficacy, it is used in every division of anesthesia.[19]

Hence this study designed to compare the efficacy of intravenous lignocaine and intravenous dexmedetomidine to effectively attenuate the haemodynamic responses accompanying laryngoscopy and intubation during anaesthetic induction.

After getting approval from the hospital ethical committee, I carried out this study in the Department of Anaesthesiology, Government Mohan Kumaramangalam Medical College Hospital, Salem, during the time of November 2014 to February 2016.

AIM OF STUDY

A PROSPECTIVE RANDOMISED STUDY ABOUT TO COMPARE THE ATTENUATION OF HAEMODYNAMIC CHANGES DURING LARYNGOSCOPY AND ENDOTRACHEAL INTUBATION WITH INTRAVENOUS LIDOCAINE VERSUS INTRAVENOUS DEXMEDETOMIDINE.

ANATOMY –NERVE SUPPLY OF LARYNX

Nerve supply of the larynx is mainly from the vagus nerve through its branches Superior laryngeal nerve and the Recurrent laryngeal nerve.

The Superior laryngeal nerve arises from the middle of the inferior ganglion of the vagus, runs downwards and forwards on the superior constrictor muscle deep to internal carotid artery and reaches the middle constrictor muscle, where it divides into external laryngeal nerve and internal laryngeal nerve. External laryngeal nerve is thin, accompanies the superior thyroid artery, pierces the deep constrictor and ends by supplying the cricothyroid muscle.

Vagus nerve gives its branch right recurrent laryngeal nerve in front of the right subclavian artery, winds backwards below the artery to reach the trachea-oesophageal groove. It is related to the inferior thyroid artery in its upper part. The nerve then passes deep to the lower border of inferior constrictor muscle, and enters the larynx behind the cricothyroid joint. It supplies all the intrinsic muscles of the larynx except the cricothyroid and carries sensory fibres to the larynx below the level of vocal cords.

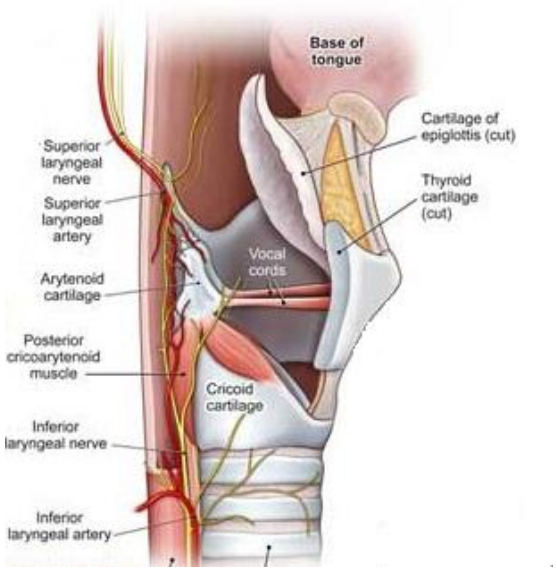

Figure 1: LATERAL VIEW OF LARYNX

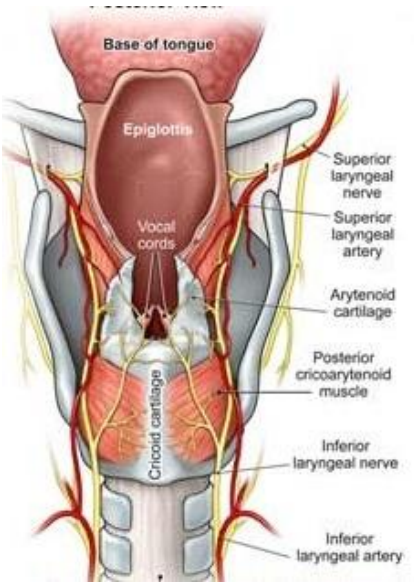

Figure 2: POSTERIOR VIEW OF LARYNX

PHYSIOLOGIC AND PATHOLOGIC RESPONSES TO LARYNGOSCOPY AND ENDO TRACHEAL INTUBATION

Intubation of trachea alters respiratory and cardiovascular physiology by both reflex response and by the physical presence of an endotracheal tube. Although the reflex responses are generally of short duration and of little significance in the majority of patients, but it is hazardous with underlying abnormalities such as hypertension, coronary artery heart disease, reactive airways and intracranial pathology.

The cardiovascular responses to laryngoscopy and intubation are bradycardia, tachycardia and hypertension. They are mediated by both the sympathetic and parasympathetic nervous systems.

Bradycardia is often seen in infants and small children during laryngoscopy and intubation. Although rarely seen in adults, this reflex is mediated by an increase in vagal tone at the sino atrial node and is virtually monosynaptic response to a noxious stimulus in the airway.[20]

The common response to laryngoscopy and endotracheal intubation is hypertension and tachycardia mediated by sympathetic efferent via the cardio accelerator and sympathetic chain ganglia. The polysynaptic nature of pathways from the IX and X nerve afferents to the sympathetic nervous

system via the brainstem and spinal cord results in a diffuse autonomic response which contains release of norepinephrine from nerve terminals and also from adrenal medulla.

Haemodynamic response due to intubation also results from stimulation of the Renin-Angiotensin system. It results in the release of renin. The effects of endotracheal intubation on the pulmonary vasculature are probably less well understood than the responses elicited in the systemic circulation. They are often coupled with changes in airway reactivity associated with intubation.

The changes in airway reactivity associated with intubation are:

1. Glottis closure reflex, i.e. laryngospasm due to brisk motor response
2. Reduction in dead space
3. Increase in airway resistance
4. Bronchospasm as a reflex response to intubation
5. Removal of the glottis barrier and may lower lung volume
6. Cough efficiency is reduced.

The balance between myocardial oxygen supply and oxygen demand should be conserved to decrease the risk of perioperative ischemia and infarction.

Factors affecting myocardial oxygen demand are

1. Basal requirements
2. Heart rate
3. Wall tension – preload and afterload
4. Contractility.

Factors affecting myocardial oxygen supply are:

Heart rate

Myocardial oxygen supply depends upon diastolic time. Hence, lower the heart rate, more the diastolic time and more the oxygen supply to myocardium.

Coronary perfusion pressure

It depends on aortic diastolic pressure and ventricular end diastolic pressure and it increases with a high aortic diastolic pressure and low ventricular end diastolic pressure.

Arterial oxygen content

Depends on arterial oxygen tension and hemoglobin concentration.

Coronary vessel diameter

Myocardial oxygen supply is directly proportional to diameter of coronary vessel. Hence when the vessel is in stenosis, the supply is reduced.

ATTENUATION OF HAEMODYNAMIC RESPONSES TO LARYNGOSCOPY AND INTUBATION

Wide range of methods were used to attenuate cardiovascular response due to direct laryngoscopy and intubation. Some of them are:

Deepening the plane of General Anaesthesia

The dosage of volatile inhalational agents required to block the responses to endotracheal intubation is termed as MAC – ei. This deep level of anaesthesia achieved by inhalational agents results in intense cardiovascular depression before endotracheal intubation. The various agents that are used to obtund the intubation responses are Halothane, Isoflurane and Sevoflurane.

Vasodilators

1. Hydralazine
2. Sodium Nitroprusside
3. Nitroglycerin

Narcotics

1. Fentanyl
2. Alfentanyl
3. Sufentanyl
4. Remifentanyl
5. Morphine
6. Pethidine

Of these, Fentanyl is the most commonly used narcotic agent to attenuate the responses to intubation.

Mechanism of action

1. Suppresses the nociceptive stimulation caused by intubation.
2. Decrease in centrally mediated sympathetic tone (Lambie et al 1974)
3. Activation of vagal tone.

Adrenergic blockers

1. α receptor blockers : Phentolamine
2. β receptor blockers : Esmolol, Metaprolol, Propranol
3. α and β receptor blockers : Labetolol

Of these, Esmolol is the most commonly used agent due to its ultrashort duration of action. It reduces resting heart rate, systolic blood pressure. There by decreases the rate pressure product, ejection fraction and cardiac index; but it maintains the coronary perfusion pressure.

Calcium Channel Blockers

1. Nifedipine,
2. Nicardipine,
3. Diltiazem,
4. Verapamil

Of these, Diltiazem is the most commonly used drug though Nicardipine has superior actions.

α2 agonist

Clonidine – it acts by suppressing the increase in sympathetic activity produced by laryngoscopy and intubation.

Midazolam

Sedative and anxiolytic

Magnesium Sulphate

Sedative and anxiolytic

Lidocaine/ lignocaine:

The various modes by which lidocaine is used to attenuate the responses to direct laryngoscopy and endotracheal intubation are:

1. Lidocaine gargles for oropharyngeal anaesthesia
2. Aerosol for intra tracheal anaesthesia
3. Topical spray over the cords
4. Regional nerve blocks
    a. Superior Laryngeal Nerve
    b. Glossopharyngeal Nerve.
5. Intravenous bolus for systemic anaesthesia

Topical anaesthesia of the upper airway proved that it less effective than intravenous lidocaine.

Intravenous form of lignocaine is easily available and most commonly used drug to attenuate laryngoscope and intubation.

Dexmedetomidine is a newer α_2 adrenergic agonist, has anesthetic sparing, analgesic, sedative, anxiolytic and sympatholytic effects. It decreases CNS sympathetic discharge in a dose-dependent manner. It has analgesic effects which is best defined as opioid sparing. In view of the fact that dexmedetomidine has shown minimal side-effects and its efficacy, it is used in every division of anesthesia.

Hence this study designed to compare the efficacy of intravenous lignocaine and intravenous dexmedetomidine to effectively attenuate the haemodynamic responses accompanying laryngoscopy and intubation during anaesthetic induction.

PHARMACOLOGY OF LIGNOCAINE

Lignocaine is one of the drug in World Health Organization essential drug list by considering its efficacy, safety and cost-effective.

CHEMISTRY:

Chemically lignocaine is 2-diethylaminoaceto-2', 6'-xylidide; $C_{14}H_{22}N_2O$. It is a stable compound, colourless in nature, crystalline solid. Lignocaine hydrochloride salt is readily soluble in water.

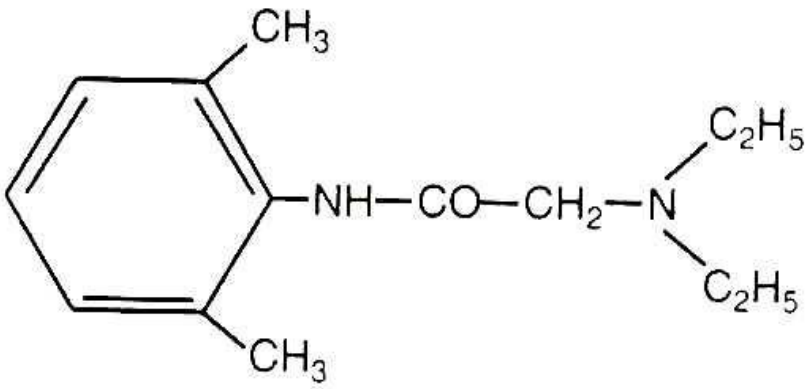

Figure 3: CHEMICAL STRUCTURE OF LIGNOCAINE

The name "xylo", or "ligno", both originally mean as "wood". The CAS number for lignocaine hydrochloride (AAN) is 6108-050.

PHARMACOLOGY

Lignocaine is a local anaesthetic drug which classified under amide group. It prevents the initiation and transmission of nerve impulses and stabilizes the neuronal membrane. It acts within the sodium channels of the nerve membrane and prevent the inward movement of sodium ions through the nerve membrane. Thereby causes a reversible block of impulse transmission along the nerve fibers. The onset of action is so rapid and the blockade may last for 1 - 1.5 hours.

Local anaesthetic drugs may have similar effects on excitable membranes in the brain and myocardium. Lignocaine is a class 1 (membrane stabilizing) antiarrhythmic agent in the heart, lignocaine reduces automaticity by decreasing the rate of diastolic (phase 4) depolarization. The duration of the action potential is shortened and the refractory period of the myocardium is reduced. The PR interval, QRS and QT durations are not affected by lignocaine.

Protein binding and Metabolism:

Protein binding varies inversely with plasma drug concentration. Between 1 and 4 mcg/ml, it is about 60-80% of drug bound with plasma protein. The acute phase reactant, alpha-1-glycoprotein also influences the drug binding. By simple passive diffusion it crosses the placenta and blood brain barrier. Lignocaine also rapidly gets distributed to all body tissues.

The half-life is 1.6 hours. The liver metabolises 80% of the lignocaine by dealkylation by cytochrome P450 and less than 10% is left unchanged in the urine. Monoethylglycinexylidide and glycine xylidide are the active metabolites. They have reduced potency but are comparable to lignocaine pharmacologically.

The metabolism of lignocaine is influenced by hepatic blood flow. So the patients with chronic liver disease, cardiac failure and after acute myocardial infarction the rate of metabolism is slow. Lignocaine and its metabolites are excreted through urine. Only less than 10% of lignocaine is excreted without being metabolized.

MECHANISM OF ACTION:

Local anesthetic blockade

Lignocaine causes reversible block of impulse generation in the nerve fibres. It binds to the sodium channels in the nerve cells, produces a conformational change which prevents the transient influx of sodium and hence decreases depolarization. As the sensory fibres are thinner, unmyelinated and can be penetrated easily they are blocked preferentially.

Antiarrhythmic effects:

It produces direct effects on purkinje fibres and acts as an antiarrhythmic. It decreases the automaticity by decreasing the slope of phase 4 and causing changes in the excitability threshold. Hence there is a reduction in the time of action potential and the refractory period. The PR interval, QRS and QT durations are not affected by lignocaine.

Anti-nociceptive effects:

By blocking the neuronal sodium channels and potassium currents, the presynaptic muscarinic and dopamine receptors it produces the anti-nociceptive effects. It also has a sodium and potassium blockade action on the spinal cord especially on the spinal dorsal neurons.

INDICATIONS:

1. In local or regional anaesthesia by
    a. Infiltration;
    b. For regional intravenous anaesthesia and
    c. Nerve blocks such as major plexus blocks and
    d. Epidural anaesthesia.
2. Treatment or prophylaxis of life-threatening ventricular arrhythmias, including those associated with myocardial infarction,
3. General anaesthesia in patients predisposed to ventricular arrhythmias, to attenuate pressor response of laryngoscopy and endotracheal intubation.
4. Digitalis intoxication, or following resuscitation from cardiac arrest.

CONTRAINDICATIONS:

1. Prior history of anaphylaxis to lignocaine or other amide-type local anaesthetics.
2. Adams- Stokes syndrome or severe degrees of SA node block, AV or intraventricular block.
3. Patients with pacemaker
4. Serious diseases of the CNS or of the spinal cord such as meningitis, spinal fluid block, cranial or spinal haemorrhage, tumors,

poliomyelitis, syphilis, tuberculosis or metastatic lesions of the spinal cord.

5. Meningitis, CSF block, ICH or spinal haemorrhage, intra cranial tumors, polio, syphilis, TB or metastatic lesions of the spinal cord.
6. Myasthenia gravis, severe shock, or impaired cardiac conduction.
7. Inflammation and sepsis in the region of the injection planned and in the existence of septicemia.
8. Epidural and spinal anaesthesia in patients with coagulation disorders or receiving anti-coagulants.

DOSE:

Lidocaine (1-1.5 mg/kg) intravenously (IV)

DRUG INTERACTIONS:

1. Anti-arrhythmic drugs:

As certain anti arrhythmic drugs (disopryramide, procainamide, mexilitene) are structurally similar to the amide type local anaesthetics, there is a possibility of augmented cardiac actions. Hence the anti-arrhythmic drugs (especially class 3) should be used cautiously with lignocaine.

2. Amiodarone:

Amiodarone decrease the clearance of lignocaine. The combination of this two drugs can decrease the threshold of convulsion, cause severe bradycardia and cardiac arrest.

3. Beta adrenoreceptor antagonists:

When propranolol and metoprolol are administered with intravenous lignocaine, the metabolism of IV administered lignocaine is reduced and hence toxic effects can appear.

4. Cimetidine:

When Cimetidine and lignocaine are administered together, the metabolism of lignocaine is decreased by cimetidine and hence toxic effects can appear.

5. Anticonvulsive agents:

Phenytoin and other antiepileptic drugs such as phenobarbitone, primidone and carbamazepine appear to enhance the metabolism of lignocaine but the

significance of this effect is not known. Phenytoin and lignocaine have additive cardiac depressant effects.

6. Inhalational anaesthetics

The minimum effective concentration of inhalational agents like nitrous oxide is reduced by lignocaine.

7. Skeletal muscle relaxants:

When lignocaine is used with suxamethonium it can cause increased duration of neuromuscular block and hence should be used carefully.

8. Structurally related local anaesthetics:

Lignocaine must use carefully in the patient getting medication structurally relate to LA.

9. Alkaline solutions:

As solubility of lignocaine decreases at pH > 7.0, with alkaline solution there is an increased tendency for precipitation.

LABORATORY TEST EFFECTS:

1. Creatinine:

N-ethylglycine, a metabolite of lignocaine interferes with enzymatic measurement of creatinine and produces values which are 15-35% greater than seen with the Jaffe method. This is seen in patients who have therapeutic levels of lignocaine.

2. Creatine kinase

Rise in creatine kinase levels are seen after intramuscular injection of lignocaine and it lasts for 48 hours and hence affects the diagnosis of myocardial infarction.

OVERDOSAGE:

CNS:

The Biphasic effects are seen as an initial stage of CNS excitation followed by depression. In the initial CNS excitation, patient manifested as dizzy or light headedness, anxiety, confusion, euphoria and convulsion. In the

later stage, CNS depression manifested as cessation of convulsions, decreased level of consciousness, respiratory depression and /or arrest.

CARDIOVASCULAR SYSTEM:

Sodium channels blockade leads to decreased depolarization of cardiac action potential in phase 0 resulting in decrease in heart rate, fall in blood pressure, cardiovascular depression and cardiac arrest.

RESPIRATORY SYSTEM:

Increased respiratory rate, respiratory depression, cessation of respiration leads to aponea can occur.

ALLERGIC REACTIONS:

Urticarial rash, oedema and anaphylaxis.

PHARMACOLOGY OF DEXMEDETOMIDINE

Dexmedetomidine has gained wide popularity as an adjunct to the anaesthetist armamentarium in India very recently. Dexmedetomidine is a selective α 2 agonist which was developed as an alternative to clonidine. The prototypical alpha 2 adrenoreceptor agonist is clonidine. dexmedetomidine is roughly eight times more specific for alpha 2 receptors when compared with clonidine, as it has a high ratio of affinity for the alpha 2 receptor (α_2/α_1 1600 : 1) matched with clonidine (α_2/α_1 200 : 1) which makes it a complete α_2 agonist.

CHEMISTRY:

Dexmedetomidine is a non-selective alpha 2 adrenoreceptor agonist, which is made up of imidazoline structure. Dexmedetomidine is the chemically active D-enantiomer of medetomidine, which has been used for sedation and analgesia in veterinary medicine for many years. It was approved for short term sedation of mechanically ventilated ICU patients by FDA.

STRUCTURE OF DEXMEDETOMIDINE

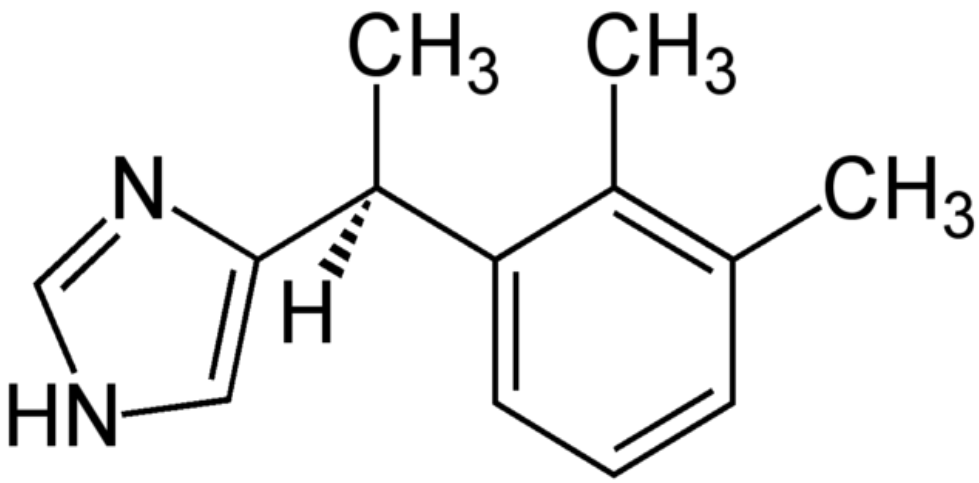

Figure 4: STRUCTURE OF DEXMEDETOMIDINE

Alpha 2 adrenoreceptor agonists have several beneficial effects like

1. Anxiolysis
2. Analgesia
3. Sedation
4. Sympatholysis

ALPHA ADRENERGIC RECEPTORS:

The adrenergic receptors are widespread in our human body. The endogenous catecholamine's like Adrenaline, Dopamine, etc act on these receptors. These receptors also mediate the clinical effects of many other drugs. A total of 9 adrenergic receptors have been synthesized so far. They include three alpha 1 adrenoreceptors, three alpha 2 adrenoreceptors and three beta adrenergic receptors.

Alpha 2 receptors have been implicated in variety of physiological functions. Three subtypes of α2 adrenoreceptors are α2A, α2B, α2C. The α2A adrenoreceptors are present in the periphery where as α2B and α2C adrenoreceptors are present in the brain and spinal cord. Postsynaptic α2 adrenoreceptors produce vasoconstriction whereas presynaptic α2 adrenoreceptors inhibit the release of norephinephrine, potentially attenuating the vasoconstriction.

The α 2 adrenoreceptor subtype is predominantly discovered in brain which mediates functions like sedation, anxiolysis, analgesia and also in behavioural changes.

When α2B adrenoreceptors are stimulated it causes vasoconstriction and systemic hypertension. The α2A receptors are responsible for causing

hypotension. Hypothermia and behavioral changes are produced by alpha 2c subtype. Thus the action of dexmedetomidine is varied with no specificity to the particular alpha 2 receptors and it has a wide variety of actions.

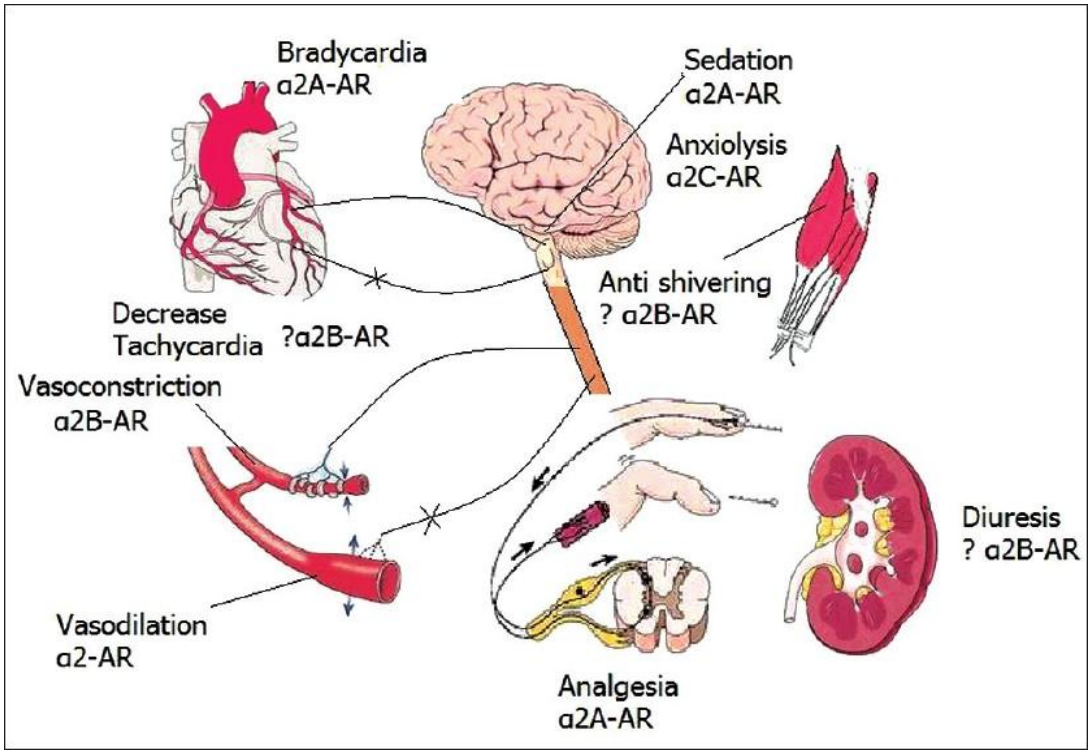

Figure 5: Action of alpha 2 adrenergic receptors

EFFECTS ON THE CENTRAL NERVOUS SYSTEM:

SEDATION:

“The alpha 2 agonists acts via the endogenous sleep endorsing pathways and employ their sedative effect. The patients remain sedated if they are not stimulated and when they are stimulated they become arousable, alert and able to respond without being uncomfortable.

They also quickly return to their sleep-like state and hence allowing the "daily wake up" tests to be conducted safely. There is limited risk for respiratory depression and is considered safe.

ANALGESIA:

The analgesic action of dexmedetomidine are multifaceted. The principle site of analgesic action of dexmedetomidine is in the spinal cord. The analgesic effects of dexmedetomidine are present, even when the drug is given intrathecally or through epidural route. Dexmedetomidine shows no action on opioid receptors. When dexmedetomidine given in the post-operative patients, the requirements of opioids were decreased by 50% and hence dexmedetomidine is considered as a narcotic sparing agent. Dexmedetomidine results only moderate reduction in pain when systemically administered and requires addition of opioids or NSAIDS for good pain control.

EFFECTS ON THE RESPIRATORY SYSTEM

The sedative effects of dexmedetomidine reduces minute ventilation, but the ventilatory response to hypercapnia is well preserved which reflecting the similarity with the physiology of normal sleep. In dogs, it was observed that it can block bronchoconstriction caused by histamine. This is major difference from opioids which cause respiratory depression. So dexmedetomidine is used for awake fibre optic intubation.

EFFECTS ON THE CARDIOVASCULAR SYSTEM

Alpha2 agonists basically cause decrease in HR, systemic vascular resistance and indirectly decrease in myocardial contraction, stroke volume and mean arterial pressure.

There is bi-phasic response seen in cardiovascular system when the bolus dose of dexmedetomidine given. In the initial phase there rise in mean arterial pressure and fall in the heart rate from baseline after 5 minutes of bolus dose. Initial phase occurs due to its action on alpha 2 receptors in the periphery. Periphery alpha 2 receptors present in the blood vessel wall leads to vasoconstriction. This initial phase can be significantly avoided by

slow infusion over 10 min. In our study, this effect was avoided by infusion of the dexmedetomidine over ten minutes.

In the later phase the heart rate comes to its baseline by 15 minutes and blood pressure drops by 15% below baseline by 1 hour. The incidence of hypotension and bradycardia may be related to the administration of a loading dose. Giving the loading dose over 10 minutes also minimizes transient hypertension.

MECHANISM OF ACTION:

Alpha 2 receptors belong to the G protein coupled receptors. They are made up of 7 transmembrane helices. By coupling uncoupling mechanisms the physiological response is produced. The proposed mechanisms include decrease of adenylyl cyclase which inhibits opening of the voltage gated calcium channels causing hyperpolarization.

DOSE:

Dexmedetomidine is usually available in 2 ml containing 100 mcg/ml. It is usually given as an loading dose of 0.5mcg- 1mcg/kg over 10 min followed by maintenance of 0.2-0.7 microgram/kg. The infusion is prepared by

adding 48 ml of 0.9% sodium chloride with 2 ml of dexmedetomidine thus making 50 ml, so that each ml contains 2mcg of dexmedetomidine.

DISTRIBUTION, METABOLISM AND ELIMINATION:

Dexmedetomidine is 95 % protein bound but does not displace any drug nor does it get displaced by any other drug. It is metabolized in the liver by glucuronidation and cytochrome P-450 enzymes .Dose adjustment will need to be made in patients with liver impairment.

It is eliminated by the kidney. No dose change needs to be made in patients with renal dysfunction.

DRUG INTERACTIONS:

Drug interactions of dexmedetomidine are many. The serum level of dexmedetomidine is increased by CYP2A6 inhibitors such as isoniazid, methosxsalen and micaonazole. Thus increasing the action of dexmedetomidine. On the other hand it increases the level of CYP2D6 substrates like tricyclic antidepressants, beta blockers, lignocaine, etc. Side effects of dexmedetomidine like bradycardia and hypotension may be increased by vasodilators and beta blockers.

DURATION OF ACTION:

The half life of dexmedetomidine is 6 minutes. The elimination half-life is about 2- 3hours.The context sensitive half time is 4 minutes after a 10 minutes of loading dose. It increased to 250 minutes after an 8 hour infusion.

ADVANTAGES:

Dexmedetomidine is a sedative and analgesic, produces sympatholysis without any respiratory depression. This is useful particularly in patients where sedation is required but depression of ventilation is not desirable. It is also an antisialogogue.

CLINICAL USES

1. ICU SEDATION:

Dexmedetomidine is used as an infusion in ICU patients for sedation and analgesia. It does not cause respiratory depression but cause moderate reduction in blood pressure and heart rate. Severe ventricular dysfunction and advanced heart block should be dealt cautiously.

2. REGIONAL ANAESTHESIA:

Dexmedetomidine is administered in dose of 0.2 mcg/kg/hr along with infusion of Remifentanil and propofol as adjunct to placement and management of regional anaesthesia including nerve blocks.

Respiration is preserved with usual dose along with hemodynamic stability without the need of additional opioids. The depth of analgesia is increased and the additional need of analgesic supplements is drastically reduced when dexmedetomidine is used in regional anaesthesia

3. AWAKE FIBREOPTIC INTUBATION:

Patient's cooperation is a vital parameter while performing awake fibre optic intubation. At the same time spontaneous respiration should be maintained throughout the procedure. The discovery of dexmedetomidine is real boon for these type of procedures where access to the anaesthetist is very limited. Dexmedetomidine causes preservation of spontaneous breathing and also sympatholysis. These benefits have been exploited during fibreoptic intubation. There has been also reports that

dexmedetomidine reduces secretions of oral airway which also supplements to the need of awake intubation.

Along with small doses of opioids and benzodiazepines with minimal dose of dexmedetomidine is used for awake intubation. Upper airway blocks can be supplemented to this to increase the plane of anaesthesia. The preservation of spontaneous respiration, sympatholysis, sedation, and analgesia of dexmedetomidine makes it a better alternative for many other drugs.

4. TOTAL INTRAVENOUS ANAESTHESIA

Dexmedetomidine is used as the main TIVA on the airway where spontaneous ventilation needs to be maintained, access to airway may not be easily possible and rapid awakening may be required.

5. MONITORED ANAESTHESIA CARE

Dexmedetomidine can also be used to provide sedation for procedures done under monitored anaesthesia care where access to airway may not be easily possible and rapid awakening may be required.

6. CARDIOTHORACIC SURGERY

Dexmedetomidine is being used as supplement to anesthetic in patients undergoing cardiovascular and thoracic procedures. It is also being used as an alternative to thoracic epidural in patients in whom it is contraindicated.

7. NEUROSURGERY

In Neurosurgery, intermittent deep and superficial plane of anesthesia is required during awake craniotomy surgeries. These are all provided by dexmedetomidine. Lack of delayed emergence, better hemodynamics and hypotensive anesthesia are all provided by dexmedetomidine. It can cause decreased blood flow to intracranial cavity. It can also used for surgeries of spinal cord.

8. MEDIASTINAL MASS

Mediastinal biopsies and radiotherapy are done under sedation with the help of dexmedetomidine. The loading dose is used as 1 mcg/kg/hr for 10 min and then 0.2-0.7 mcg/kg/hr maintenance infusion. Dexmedetomidine is safe as it does not depress respiration or the cardiovascular system and yet provides the required sedation.

CONTRAINDICATION:

1. Infusion over 24 hours
2. In obstetric patients (safety not known)
3. Conditions causing bradycardia and heart block.
4. Left ventricular failure (ejection fraction less than 30%).
5. Hypovolemic and hypotensive conditions
6. Increased intra cranial tension

ANTIDOTE

Atipamezole, the alpha 2- adrenoreceptor antagonist reverses all the actions exerted by dexmedetomidine.

REVIEW OF LITERATURE

REID & BRACE (1940) postulated that reflex circulatory responses to laryngeal instrumentation were mediated through the vagus nerve & they named it as "vaso vagal reflex".

KING et al (1951) used deep anaesthesia to abolish the reflex circulatory response to tracheal intubation.[21]

WYCOFF C.C. (1960) in his study stated that topical anaesthesia of the pharynx along with superior laryngeal nerve blocks can decreased the rise in mean blood pressure caused by laryngoscopy.[22]

STEIMHANS & GASKIN (1963) found that intravenous lignocaine suppressed the cough reflex. It is very easy to predict that if the cough is suppressed, the rise in blood pressure, pulse rate and intracranial pressure noticed on laryngeal instrumentation would be blunted by this technique.[23]

FORBES & DALLY (1970) observed that laryngoscopy & endotracheal intubation were immediately associated with an average increase in mean arterial pressure of 25mmHg in all 22 normotensive

patients. These responses were interpreted as due to reflex adrenal stimulation.[24]

MASSON & ECKANKOFF (1971) proved that the hypertensive response in patients can be significantly decreased by simple lignocaine spray.

PRYS ROBERTS et al (1971) found that the increase in heart rate & blood pressure are much more exaggerated in hypertensive patients.[25]

DENLINGER.J.K. & ELLISON.N.E. (1974) have used intratracheal lignocaine spray which causes a 50% reduction in the hypertensive response.[26]

VICTORIA FARIA BLANC & NORMAND.A.G. (1974) in their article of "complications of tracheal intubation have classified the neurogenic or reflexly mediated complications into 3 different categories.

Laryngo Vagal Reflexes – which gives rise to laryngospasm, bronchospasm, apnoea, bradycardia, cardiac dysrhythmias & arterial hypotension. The mere presence of the tracheal tube seem to be the most common cause of bronchospasm in anaesthetized asthmatic patients.

Laryngo Sympathetic Reflexes – which include tachycardia, tachyarrythmia & acute rise in mean blood pressure are occurred as common complications. The hypertensive and hyperdynamic state occur after intubation may be related to sudden rise noradrenaline with respect to overall catecholamines.

Laryngo Spinal Reflexes – which include coughing, vomiting & bucking.

TAMMISTO.T. & AROMAA.U. (1977) has shown in his study that tolerance to the endotracheal tube is more rationally achieved by small doses of narcotic analgesics (e.g. fentanyl 0.5 to 1 microgram/kg) than by increasing the dose of thiopentone.[27]

McCAMMON RL et al (1981) showed in their study that intravenous Propanolol does not confirm the attenuation of increases in HR & MAP associated with laryngoscopy & tracheal intubation.[28]

FASSOULAKI A, KANIARIS P (1983) proved in their study that intranasal administration of nitroglycerine decreases the hemodynamic response to laryngoscopy & tracheal intubation.[29]

TAM.S. et al (1985) found attenuation of circulatory responses to laryngoscopy & before intubation.[30]

BATRA Y.K., INDU.B., PURI.G.D. (1988) observed that oral clonidine attenuated the PR & BP response to laryngoscopy & tracheal intubation.[31]

NISHIKAWA T., NAMIKI A (1989) proved in their study that intravenous Verapamil attenuates the pressor response to laryngoscopy & tracheal intubation.[32]

HATANO, IMAI, KOMATSU, MORI K.(1989) showed in their study that intravenous administration of isosorbide dinitrate attenuates the pressor response to laryngoscopy & tracheal intubation.[33]

T.NISHINO.K., HIRAGA & K.SUGIMORI (1990) proved that lignocaine had a dose dependent effect on the extubation reflex, cough reflex in patients anaesthetized with enflurane & that 1.5mg/kg of intravenous lignocaine can suppress the cough reflex & other reflexes during intubation, extubation, bronchoscopy & laryngoscopy when duration of these procedures is relatively brief.[34]

LICKER M, FARINELLI C, KLOPFENSTEIN CE (1995) concluded in their study that thoracic epidural blockade combined with general anaesthesia was associated with preserved baroreflex function and

it afforded haemodynamic protection during laryngoscopy and intubation in the elderly.[35]

KITAMURA T, YAMADA Y, CHINZEI M, DU HL, HANAOKA K (2001) showed in their study that using the new intubation device Styletscope helps in the attenuation of hemodynamic responses to tracheal intubation.[36]

YOO KY, JEONG ST, HA IH, LEE J (2003) showed that nitrous oxide reduces pressor but augments norepinephrine response to laryngoscopy and endotracheal intubation.[37]

SPLINTER.W.M., CERVENKO.F. (1989) has concluded in their study both lignocaine and fentanyl are recommended adjuncts to induction of anaesthesia with thiopentone in geriatric patients.

Tarn, Stanley; Chung, Frances; Campbell, Michael. et al (38) found that Intravenous lidocaine at the dosage of 1.5 mg/kg attenuates rise in heart rate and Blood Pressure only when given **3**min before intubation, and offers no protection against post-intubation haemodynamic response when given at 1, 2, or 5 minutes prior to intubation.[38]

Abou-Madi et al in 1977 studied circulatory response to laryngoscopy and tracheal intubation after small (0.75 mg/kg) and large (1.5 mg/kg) iv doses of lidocaine in eighty ASA grade 1 to 4 patients and found that the larger dose was more efficient in attenuating circulatory responses to tracheal intubation.[39]

M A Kaleem Siddiqui et al 2015 found out that intravenous lignocaine is less effective to attenuate the pressor response and a better alternative should be used after further comparative studies.[40]

R.Saraf, M.Jha, Sunil Kumar.V, K. Damani, S.Bokil, D. Galante 2013 et al found Dexmedetomidine at a dose of 0.6µg/kg in 10 ml NS, given 10 min before induction significantly obtunds the haemodynamic response to laryngoscopy and tracheal intubation in adult and paediatric patients. It also decreases the requirement of induction dose of thiopentone and also the requirement of the total dose of vecuronium bromide for muscle relaxation without significant side effects.[41]

Xuexin Feng, Weixiu Yuan et al (2014) stated that a single dose of dexmedetomidine given prior to induction of general anaesthesia significantly decreased the stress hormone response to endotracheal

intubation, kept haemodynamics more stable, and contributed to perioperative safety.

Dr. Sagar Gandhi et al (2014) concluded that DEXMEDETOMIDINE when used as Intravenous premedicant in the dosage of 0.6 μg/kg gives favorable effect in attenuation of circulatory response to laryngoscopy and endotracheal intubation as compare to FENTANYL in dose of 2 μg/kg.[42]

Varshali M Keniya, Sushma Ladi, Ramesh Naphade et al 2011 stated that Dexmedetomidine can used as a pre-medication in general anaesthesia. It reduces the intraoperative anaesthetic drug dosage. It has opioid and anaesthetic sparing property. It blunts the circulatory response to tracheal intubation. It has no adverse effect on cardiovascular and respiratory system even after continuous infusion in intra operative period.[43]

Srivastava VK, Agrawal S, Gautam SS, Ahmed M, Sharma S, Kumar R et al 2015 stated that use of dexmedetomidine for attenuation of hypertensive response to laryngoscopy and intubation is more effective than esmolol in preventing such hemodynamic responses in neurosurgical patients.[44]

Michell Gulabani et al 2015 conclude that dexmedetomidine in the dosage of 1 μg/kg over 10 min before induction of anaesthesia efficiently attenuates the haemodynamic changes to laryngoscopy and intubation.[45]

Dexmedetomidine administered in a dose of 0.5μg/kg over 10 min before induction of anaesthesia was effective in controlling the tachycardia but incompletely attenuated the rise in SBP and DBP.

Further, lignocaine in the dosage of 1.5 mg/kg given 3 minutes before laryngoscopy and intubation was more effective than dexmedetomidine 0.5 μg/kg in attenuating the rise in systolic and diastolic blood pressure at 3 min and 5 min after endotracheal intubation.

MATERIALS AND METHOD

Type of Study

A prospective, randomised double blind study

Place of study

Operation Theatre in Govt. Mohan Kumaramangalam medical College and Hospital.

Subjects

61 patients in the age group **18-60 of ASA PS I or II**.

Materials

Philips MP 40 monitor, Dexmedetomidine ampoules, Lignocaine vial, Sevoflurane.

It was conducted after approval from Ethics Committee of institution. The study was explained to all the patient and written informed consent from each patients.

The study compared the efficiency of lignocaine and dexmedetomidine for attenuation of the haemodynamic changes happened during laryngoscopy and intubation in elective patients requiring general anaesthesia.

INCLUSION CRITERIA

1. Patients in American Society of Anesthesiologists
    a. Physical status class I
    b. Physical status class II
2. Patients with modified Mallampatti scores I & II
3. Age **18-55**years

EXCLUSION CRITERIA

1. Patients in American Society of Anesthesiologists
    a. physical status class III
    b. physical status class IV
2. Patients with modified Mallampatti scores III & IV
3. Patients with predicted difficult airway
4. Obese patients

5. Patients with Systemic Hypertension, CAD, H/O Cerebrovascular Accidents, CRF, Valvular Heart Diseases, patients on antihypertensives or cardiac drugs
6. Patients posted for emergency surgeries
7. Patients with full stomach
8. If the intubation time has exceeded 15 seconds
9. Age <20 and >55 years
10. Patient undergoing procedures requiring head & neck manipulation

ASSESSMENT

All patients were assessed by a detailed physical examination supported by investigations like routine blood tests- Hb, blood sugar, blood urea, serum creatinine, serum electrolytes, chest X ray PA view, Electrocardiogram, etc.

RANDOMIZATION:

The patients were randomly allocated to two groups of 30 with the help of a computer generated table of random numbers to receive following drugs:

Group L: 30 patients were given inj Lignocaine 1.5 mg/kg body weight intravenously, 3 minutes before intubation.

Group II: 30 patients were given Inj Dexmedetomidine (1 mcg/kg body weight) intravenously over 10 minutes, given 10 minute before intubation.

ANAESTHESIA PROTOCOL

Preoperative visit was made to allay anxiety, and a good rapport was established with the patient.

PREMEDICATION

All patients were given Tab Diazepam 10 mg orally the night before surgery. Patients were kept nil orally 6-8 hours prior to surgery. Now patients were randomly divided by computer into two groups. In preoperative room baseline parameters were observed and documented. All patients were given Inj Glycopyrolate 10 mcg/kg body weight intramuscularly 45 minutes before surge.

MONITORING

Patients shifted to the operating room, an 18-gauge intravenous cannula was inserted in the forearm and infusion of ringer lactate was started. Standard multimonitor was connected - ECG, NIBP and pulse oximeter. NIBP was recorded every two minutes.

INDUCTION AND INTUBATION:

Patients were given inj. fentanyl 2mic.gm/kg body wt. Group D received 1 mcg/kg of Inj. Dexmedetomidine in 10 ml of normal saline over 10 minutes and 5ml of normal saline 3 minutes before induction. Group L received 10 ml of normal saline over 10 minutes and Inj. Lignocaine 1.5 mg/kg diluted in 5 ml of normal saline 3 minutes before induction. These solutions of 10 ml and 5 ml were prepared by first anaesthesiologist.

The second anaesthesiologist, who was not aware of the groups, administered the drug and monitored the patients recording vital parameters before intubation and immediatly after intubation and also 1min, 3min or 5 min after laryngoscopy and endotracheal intubation according to the group to which they were assigned.

The laryngoscopy and intubation were performed by the third anaesthesiologist who was also blinded to the drug given. Patients were preoxygenated for 5minutes. Patients were induced with Inj. Propofol 2 mg iv and followed by Inj. Suxamethonium 2mg iv. Thereafter all the patients were manually ventilated with bag and mask with 100% oxygen for 3minutes. Laryngoscopy and intubation was then done and the time taken for the same was noted.

Those that took > 15 seconds were excluded from the study. After confirming the position of the endotracheal tube, anaesthesia was maintained for the next 5 minutes with 67% nitrogen and 33% oxygen. No surgical stimulation was permitted for 5minutes after intubation. The baseline, before intubation, immediately after intubation (0 minutes), 1 minute, 3 minutes and 5 minutes after intubation values of circulatory variables such as HR, SBP, DBP and MBP were recorded.

RESULTS

TABLE: 1 DESCRIPTIVE ANALYSIS OF AGE GROUP IN STUDY GROUP (N=61)

Age Group	Frequency	Percentage
< 20 Years	3	4.92%
20 - 39 Years	30	49.18%
40 - 59 Years	26	42.62%
> 60 Years	2	3.28%
Total	61	100.00%

Out of 61 subjects,

3 (4.92%) were with the age less than 20 years,

30 (49.18%) subjects were in the age group 20 to 39 years,

26 (42.62%) were in age group 40 to 59 years and

2 (3.28%) were with the age less than 60.

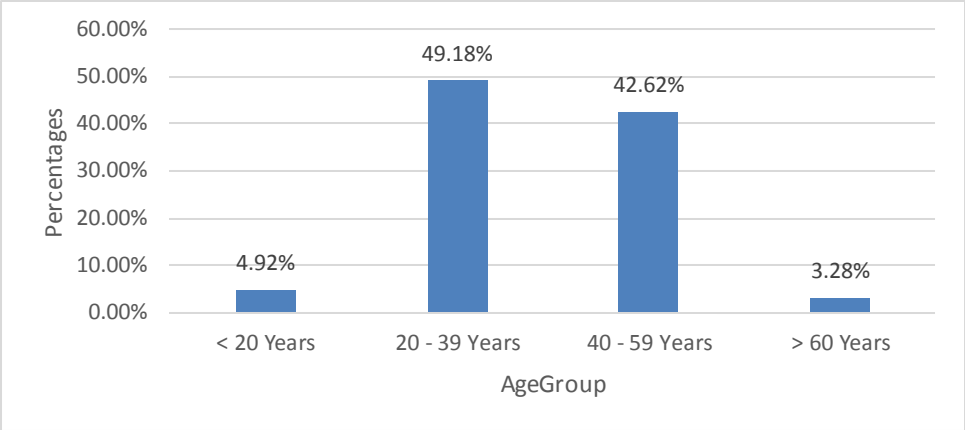

CHART: 1 BAR CHART OF AGE GROUP DISTRIBUTION IN STUDY GROUP (N=61).

TABLE: 2 DESCRIPTIVE ANALYSIS OF SEX IN STUDY GROUP (N=61)

SEX	Frequency	Percentage
Female	14	22.95%
Male	47	77.05%
Total	61	100.00%

Out of 61 people, 14 (22.95%) were females and 47 (77.05%) were males.

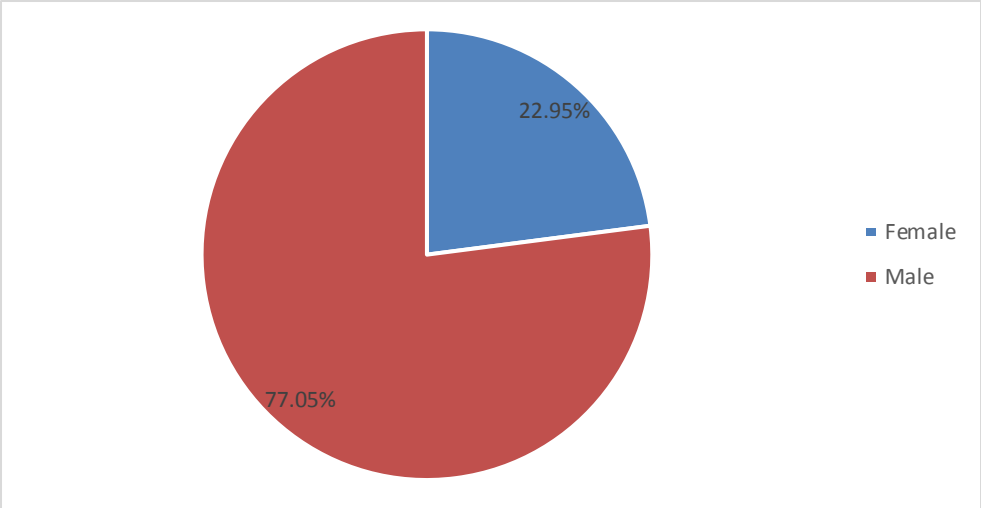

CHART: 2 PIE CHART OF SEX DISTRIBUTION IN STUDY GROUP (N=61)

TABLE: 3 DESCRIPTIVE ANALYSIS OF GROUP IN STUDY GROUP (N=61)

GROUP	Frequency	Percentage
Dexmedetomidine	30	49.18%
Lignocaine	31	50.82%
Total	61	100.00%

In the group of 61, 30 (49.18%) are in the Dexmedetomidine group and 31 (50.82%) are in the Lignocaine group.

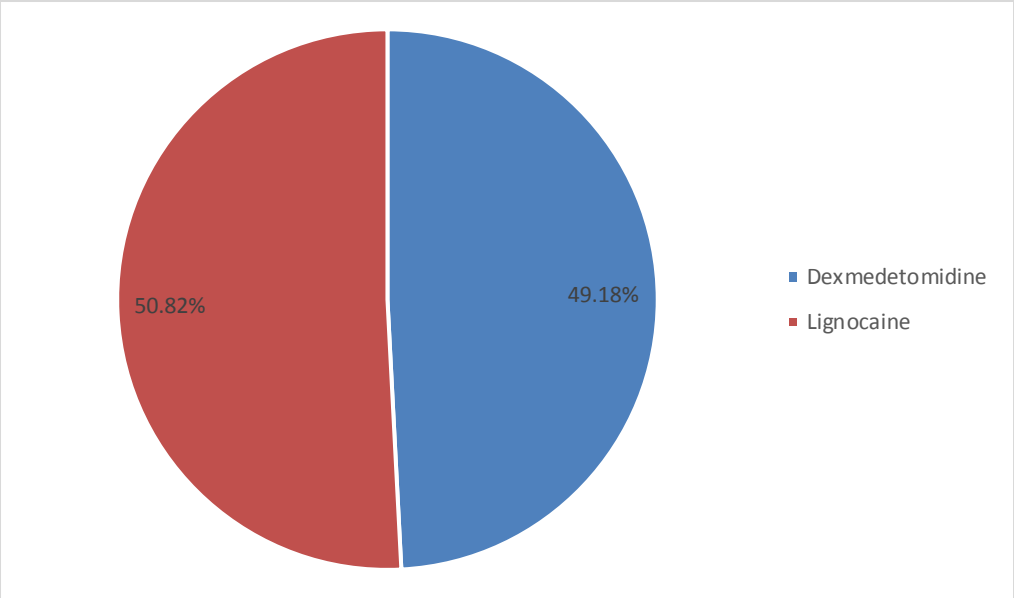

CHART: 3 PIE CHART OF GROUP DISTRIBUTION IN STUDY GROUP (N=61)

TABLE: 4 CROSS TAB OF SEX AND AGE GROUP.

Parameter	GROUP		P value
	Dexmedetomidine	Lignocaine	
SEX			
Female	6	8	3.271
	20.00%	25.81%	
Male	24	23	
	80.00%	74.19%	

Parameter	GROUP		P value
	Dexmedetomidine	Lignocaine	
Age Group			
< 20 Years	2	1	0.325
	6.67%	3.23%	
20 - 39 Years	17	13	
	56.67%	41.94%	
40 - 59 Years	11	15	
	36.67%	48.39%	
> 60 Years	0	2	
	0.00%	6.45%	

In the study group of 61 persons, 6 (20.0%) females were in the group Dexmedetomidine and 8 (25.81%) females were in the group Lignocaine. 24 (80.00%) males were in the group Dexmedetomidine and 23 (74.19%) were in the group Lignocaine.

Out of 61 subjects, 3 subjects were under 20 years with 2 (6.67%) subjects in the group Dexmedetomidine, and 1 (3.23%) subjects in the group Lignocaine. 30 subjects were in the age group 20 to 39 years with 17 (56.67%) in the group Dexmedetomidine and 13 (41.94%) subjects in the group Lignocaine. 26 subjects were in the age group 40 to 59 years with 11(36.67&) in the group Dexmedetomidine and 15 (48.39%) subjects were

in the group Lignocaine. 2 subjects were with the age more than 60 and 2 (6.45%) were in the group Lignocaine.

TABLE: 5 Independent Sample T Test of Parameters WEIGHT, HEIGHT, BMI, BHR, BSBP, BDBP, BMBP.

Parameter	Mean ± STD		P-value
	Lignocaine	Dexmedetomidine	
WEIGHT	66.29 ± 7.528	68.9 ± 4.474	0.107
HEIGHT	167.00 ± 6.503	168.1 ± 5.797	0.528
BMI	23.67 ± 1.605	24.40 ± 1.424	0.068
BHR	84.48 ± 11.16	85.86 ± 9.5	0.604
BSBP	117.4 ± 13.34	118.9 ± 11.28	0.656
BDBP	72.19 ± 8.79	72.86 ± 7.3	0.746
BMBP	87.32 ± 10.27	88.2 ± 8.54	0.718

The mean value of weight in the group Lignocaine is 66.29 and in the group Dexmedetomidine is 68.9 and its association is stastically not significant. The mean value of height in the group Lignocaine is 167 and in the group Dexmedetomidine the mean value of height is 168.1 and association is stastically not significant.

The mean value of BMI in the groups Lignocaine and Dexmedetomidine were 23.67 and 24.40 respectively and its p value is stastically not significant.

The mean values of BHR in the groups Lignocaine and Dexmedetomidine were 84.48 and 85.86 respectively and p value stastically non-significant.

The mean of BSBP in the group Lignocaine is 117.4 and in the group Dexmedetomidine is 118.9 with p-value stastically not significant.

The mean of BDBP in the group Lignocaine is 72.19 and in the group Dexmedetomidine is 72.86 with p-value stastically significant.

The mean value of BMBP in the groups Lignocaine and Dexmedetomidine were 87.32 and 88.2 respectively and its p value is stastically not significant.

Hence, the study groups are comparable.

TABLE: 6 Independent Sample T Test of Parameters PLHR, PLSBP, PLDBP, PLMBP.

Parameter	Mean ± STD		P value
	Lignocaine	Dexmedetomidine	
PLHR	82.35 ± 11.19	75.76 ± 8.46	0.0121
PLSBP	107.9 ± 11.64	101.9 ± 5.19	0.0122
PLDBP	65.64 ± 6.12	60.86 ± 3.69	<0.0001
PLMBP	79.8 ± 7.73	74.53 ± 4.17	<0.0001

- The mean of PLHR in the group Lignocaine is 82.35 and 75.76 in the group Dexmedetomidine with the association stastically significant.
- The mean of PLSBP in the groups Lignocaine, Dexmedetomidine were 107.9, 101.9 respectively with p value stastically significant.

- The mean of PLDBP in the groups Lignocaine and Dexmedetomidine were 65.64 and 60.86 respectively with association statistically significant.
- The mean of PLMBP of the groups Lignocaine & Dexmedetomidine were 79.8 and 74.53 respectively with association statistically significant.
- This difference in the pre laryngoscopy value is due to alpha two agoinst action of dexmedetomidine, whereas lignocaine has membrane stabilizing action.

TABLE: 7 Independent Sample T Test of Heart Rate

Parameter	Mean±STD		P value
	Lignocaine	Dexmedetomidine	
BHR	84.48 ± 11.16	85.86 ± 9.5	0.6048
PLHR	82.35 ± 11.19	75.76 ± 8.46	0.0121
0-Heart Rate	86.16 ± 11.52	71.5 ± 7.46	<0.0001
1-Heart Rate	82.19 ± 9.74	66.1 ± 6.143	<0.0001
3-Heart Rate	80.70 ± 8.691	62.06 ± 5.13	<0.0001
5-Heart Rate	79.09 ± 7.828	56.8 ± 4.080	<0.0001

The mean values of BHR in the groups Lignocaine and Dexmedetomidine were 84.48 and 85.86 respectively and p value stastically non-significant. The mean of PLHR in the group Lignocaine is 82.35 and 75.76 in the group Dexmedetomidine with the association stastically significant. The mean of the heart rate at 0 minute in the group Lignocaine was 86.16 and in the

group Dexmedetomidine was 71.5 and the association with stastically significant.

The mean of the heart rate at 1 minutes in the groups Lignocaine and Dexmedetomidine were 82.19 and 66.1 respectively and p value is stastically significant. The mean of the heart rate at 3 minutes in the group Lignocaine was 80.70 and in the group Dexmedetomidine was 62.06 and the association with stastically significant. The mean of the heart rate at 5 minutes in the groups Lignocaine & Dexmedetomidine were 79.09 & 56.8 respectively and p value is stastically significant.

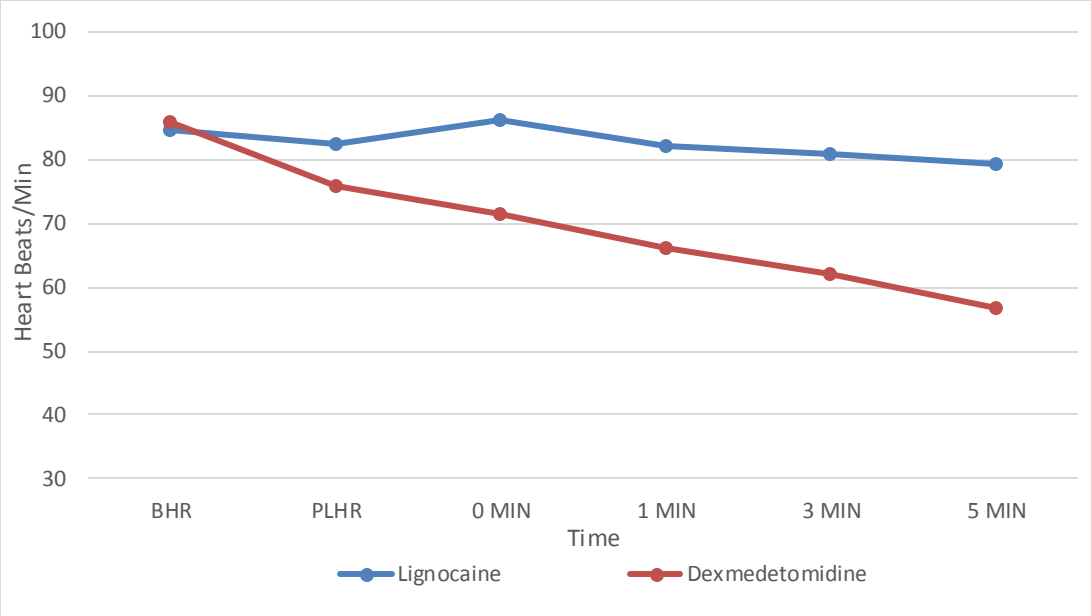

CHART: 4 TRENDS IN HEART RATE

TABLE: 8 Independent Sample T Test of Systolic Blood Pressure

Parameter	Mean ± STD		p value
	Lignocaine	Dexmedetomidine	
BSPB	117.4 ± 13.34	118.9 ± 11.28	0.6565
PLSPB	107.9 ± 11.64	101.9 ± 5.19	0.0122
0-SBP	112.4 ± 7.29	101.1 ± 3.98	<0.0001
1-SBP	105 ± 6.76	96.2 ± 3.54	<0.0001
3-SBP	98.25 ± 6.37	92.36 ± 3.25	<0.0001
5-SBP	95.83 ± 4.194	88.86 ± 4.342	<0.0001

The mean of BSBP in the group Lignocaine is 117.4 and in the group Dexmedetomidine is 118.9 with p-value stastically not significant. The mean of PLSBP in the groups Lignocaine and Dexmedetomidine were 107.9 and 101.9 respectively with p value stastically significant. The mean of the Systolic Blood Pressure at 0 minute in the group Lignocaine was 112.4 and in the group Dexmedetomidine was 101.1 and the association with stastically significant. The mean of the Systolic Blood Pressure at 1

minutes in the groups Lignocaine & Dexmedetomidine were 105 and 96.2 respectively and p value is stastically significant. The mean of the Systolic Blood Pressure at 3 minutes in the group Lignocaine was 98.25 and in the group Dexmedetomidine was 92.36 and the association with stastically significant. The mean of the Systolic Blood Pressure at 5 minutes in the groups Lignocaine & Dexmedetomidine were 95.83 & 88.86 respectively and p value is stastically significant.

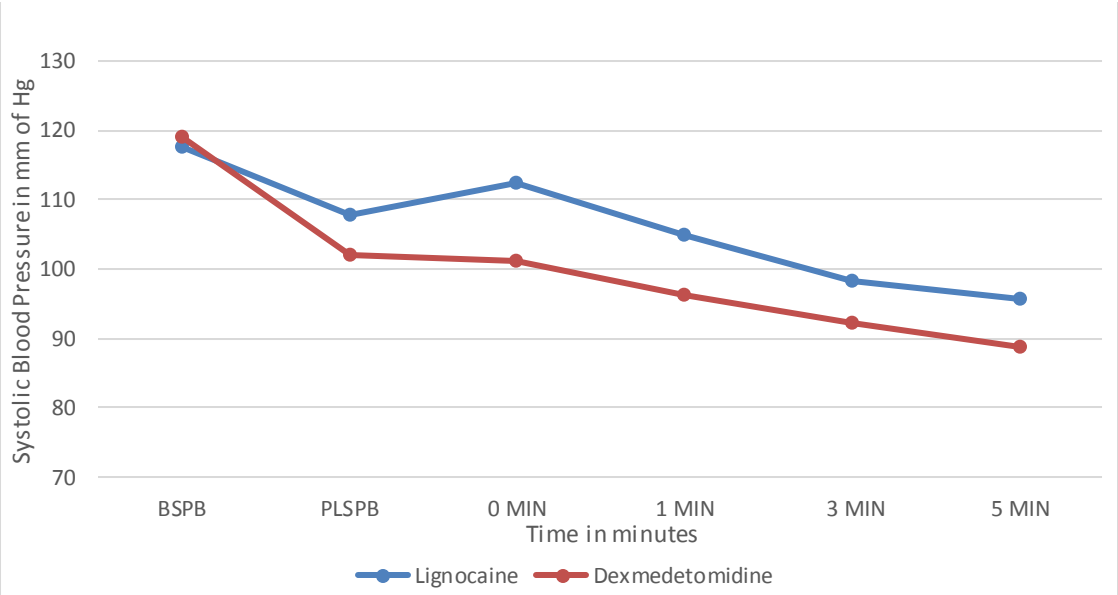

CHART: 5 TRENDS IN SBP

TABLE: 9 Independent Sample T Test of Diastolic Blood Pressure

Parameter	Mean ± STD		p value
	Lignocaine	Dexmedetomidine	
BDBP	72.19 ± 8.79	72.86 ± 7.3	0.7466
PLDBP	65.64 ± 6.12	60.86 ± 3.69	<0.0001
0-DBP	66.67 ± 4.55	60.5 ± 2.73	<0.0001
1-DBP	62.38 ± 5.84	54.46 ± 2.85	<0.0001
3-DBP	58.03 ± 5.55	48.93 ± 1.66	<0.0001
5-DBP	54.32 ± 4.23	45.8 ± 1.49	<0.0001

The mean of DBP in the group Lignocaine is 72.19 and in the group Dexmedetomidine is 72.86 with p-value stastically significant. The mean of PLDBP in the groups Lignocaine and Dexmedetomidine were 65.64 and 60.86 respectively with association statistically significant. The mean of the Diastolic Blood Pressure at 0 minutes in the group Lignocaine was 66.67 and in the group Dexmedetomidine was 60.5 and the association with stastically significant.

The mean of the Diastolic Blood Pressure at 1 minutes in the groups Lignocaine & Dexmedetomidine were 62.38 & 54.46 respectively and p value is stastically significant. The mean of the Diastolic Blood Pressure at 3 minutes in the group Lignocaine was 58.03 and in the group Dexmedetomidine was 48.93 and the association with stastically significant. The mean of the Diastolic Blood Pressure at 5 minutes in the groups Lignocaine, Dexmedetomidine were 54.32, 45.8 respectively and p value is stastically significant.

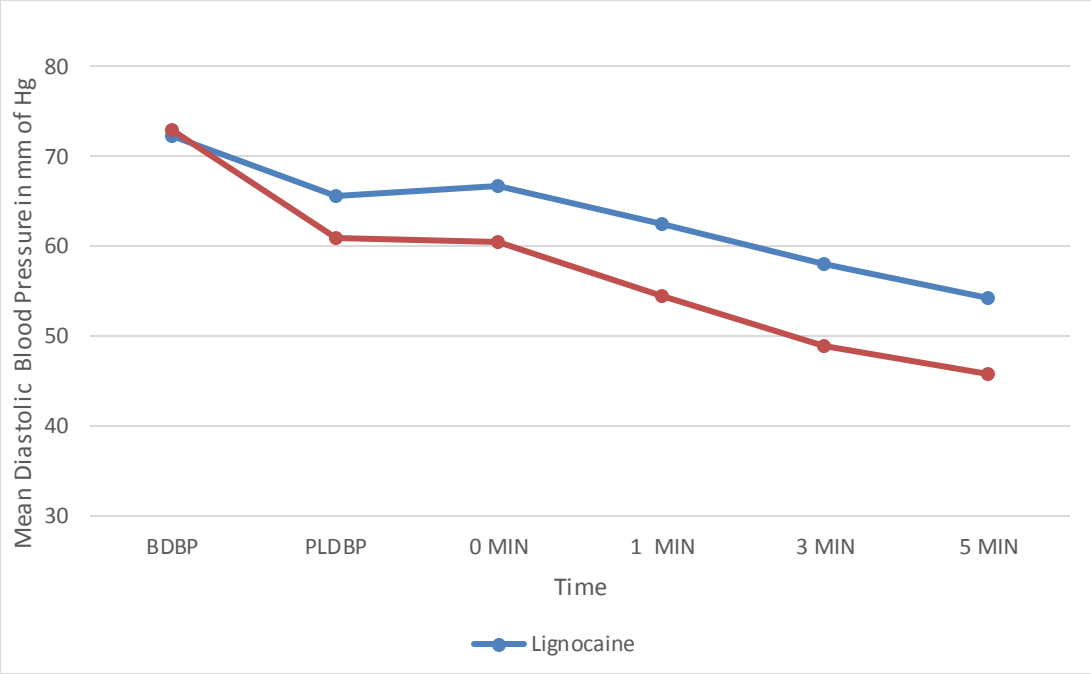

CHART: 6 TRENDS IN DBP

TABLE: 10 Independent Sample T Test of Mean Blood Pressure

Parameter	Mean ± STD		p value
	Lignocaine	Dexmedetomidine	
BMBP	87.32 ± 10.27	88.2 ± 8.54	0.7186
PLMBP	79.80 ± 7.73	74.53 ± 4.17	<0.0001
0-MBP	81.96 ± 5.27	74.1 ± 2.92	<0.0001
1-MBP	76.58 ± 5.98	68.4 ± 2.79	<0.0001
3-MBP	71.51 ± 5.61	63.43 ± 1.87	<0.0001
5-MBP	68.12 ± 4.32	60.43 ± 1.41	<0.0001

The mean value of BMBP in the groups Lignocaine and Dexmedetomidine were 87.32 and 88.2 respectively and its p value is stastically not significant. The mean of PLMBP of the groups Lignocaine and Dexmedetomidine were 79.8 and 74.53 respectively with association statistically significant. The mean of the Mean Blood Pressure at 0 hour in

the group Lignocaine was 81.96 and in the group Dexmedetomidine was 74.1 and the association with stastically significant.

The mean of the Mean Blood Pressure at 1 minutes in the groups Lignocaine & Dexmedetomidine were 76.58 and 68.4 respectively and p value is stastically significant. The mean of the Mean Blood Pressure at 3 minutes in the group Lignocaine was 71.51 and in the group Dexmedetomidine was 63.43 and the association with stastically significant. The mean of the Mean Blood Pressure at 5 minutes in the groups Lignocaine and Dexmedetomidine were 68.12 and 60.43 respectively and p value is stastically significant.

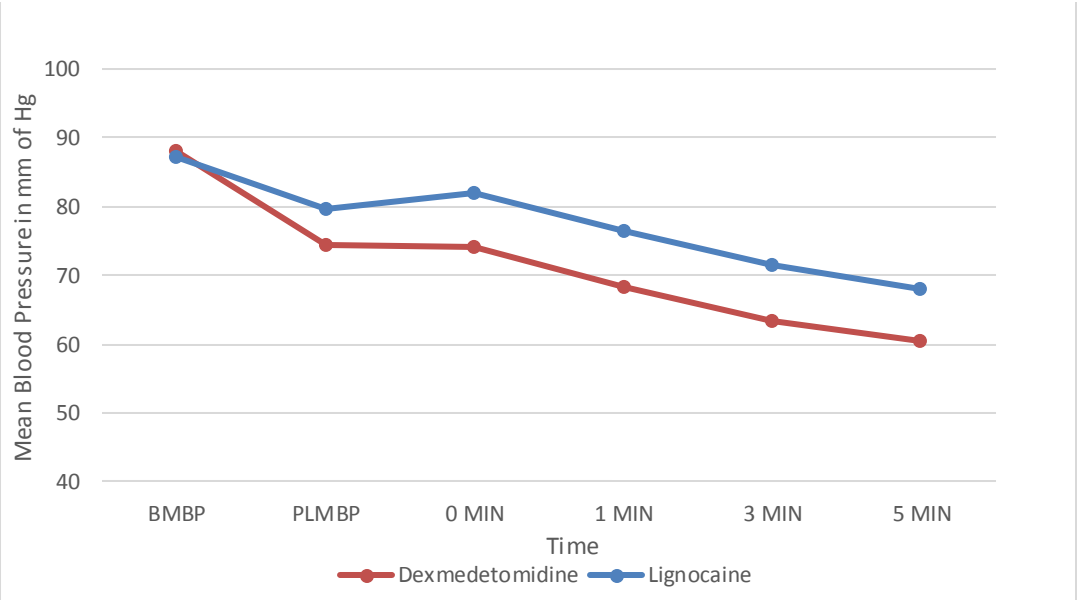

CHART: 7 TRENDS IN MBP

DISCUSSION

The hemodynamic response are characterized by tachycardia and hypertension during handling in the larynx, by means of laryngoscopy and intubation.

Stimulation of mechanoreceptors in the wall of pharynx, epiglottis and vocal cords, is thought to be the cause for this hemodynamic response. Shribman *et al* found that laryngoscopy only or followed by endotracheal intubation rises the HR, BP and catecholamine levels. These changes were reported to be greatest at 1 minute after intubation of the trachea that lasts for 5-10min. HR may rise from 26% to 66% and the SBP may rise from 36% to 45% when no precise actions are taken to avoid this hemodynamic response. It can withheld by the patients without cardiovascular and respiratory co morbidities.[46, 47]

Myocardial ischemia may happen during the laryngoscopy & intubation sequence in patients with CAD. There is a higher chances of progress of intraoperative ischemia into perioperative myocardial infarction. Interventions like direct laryngoscopy involving severe sympathetic stimuli, prevention of tachycardia, hypertension and elevated

total oxygen consumption due to sympathetic activity may prove beneficial in patients with limited cardiac reserve.

Various reports have look over the effect of lignocaine to blunt the sympathoadrenal pressure response.

Lev and Rosen in their study reviewed the use of prophylactic lignocaine as a preintubation medication.[48]

A dose of 1.5 mg/kg intravenously 3 min prior to intubation was employed and was found to be optimal for attenuating the sympathoadrenal response to laryngoscopy and intubation without any overt harmful effects. We also administered lignocaine 1.5 mg/kg 3 min before intubation in our study and observed a general decline in HR, SBP, and DBP. But there is spike in all the hemodynamic parameters HR, SBP, DBP and MBP immediately after intubation. The decrease in HR and blood pressure in our study might also be attributed to the use of anaesthetic agents such as opioids (fentanyl) and inhalational agents.

Wilson *et al*. in their study stated that IV lignocaine is beneficial in preventing the hemodynamic changes to laryngoscopy and intubation.[49] We noted the maximal decline of HR to be at 3 min after intubation from

basal value, while then maximal decline in SBP and DBP is observed at 5 min post intubation.

However this decline of HR and blood pressure can possibly be attributed to the combined effects of fentanyl and inhalational agents administered during the maintenance of anaesthesia. From our statistical analysis we also infer that though there is a general decline in HR after administration of lignocaine, but at the time interval corresponding to 0 min post-intubation, we observed an increase in HR. This shows that the pressure response was incompletely abolished by lignocaine.

Lignocaine attenuated the rise in blood pressure but not prevented it totally. The rise persisted for 1 min in the lignocaine group after intubation. In our study we found that lignocaine sufficiently attenuated the above mentioned hemodynamic response, but this attenuation was not complete and a spike in SBP was observed up to 1 min of post intubation.

We also noticed 2 spikes at 1 min and 3 min intervals post-intubation in the DBP recordings which are in concordance with the above study. In our study we also did not encounter any side effects like hypotension or bradycardia when lignocaine at a dose of 1.5 mg/kg was employed.

Recent studies however, doubted the efficacy of lignocaine. In studies by Singh *et al*. Kindler *et al* Intravenous lignocaine of dosage 1.5 mg/kg was not effective to decrease the acute haemodynamic response after intubation.[50]

In a study conducted by Pathak *et al* it was shown that lignocaine 1.5 mg/kg was not effective in blunting the responses during laryngoscopy and tracheal intubation when compared with two different doses of alfentanil (15 μg/kg and 30 μg/kg). However in our study, we used fentanyl universally in both the groups. From the interpretation of the results of our study we concluded that lignocaine attenuated but did not fully abolish the pressure response to laryngoscopy and intubation.[51]

Alpha two adrenergic agonists decreases sympathetic reflex and thereby attenuating the hemodynamic responses to laryngoscopy and intubation.[52]

They also decrease the requirement of anaesthetic drugs and therefore can be used as an adjunct in general anaesthesia. Dexmedetomidine is a highly selective and specific alpha two adrenergic agonist. Therefore, it is increasingly being used as an agent to attenuate the pressure response.[53]

Sagiroglu *et al*. concluded that the overall control of hemodynamic responses to tracheal intubation were better with dexmedetomidine 1 μg/kg as compared to dexmedetomidine 0.5 μg/kg. In our study we concluded that 1 μg/kg of dexmedetomidine significantly reduced the increase in HR associated with laryngoscopy and intubation compared with lignocaine.

In the study conducted by Sagiroglu *et al*. the results of SBP, DBP and mean arterial pressure were significantly lower in the group given dexmedetomidine 1 μg/kg .

This is in agreement with our study results, which show a statistically significant decline in systolic and DBPs in the group administered dexmedetomidine 1 μg/kg.[55]

On comparison we concluded that dexmedetomidine 1 μg/kg brought upon a maximal reduction in SBP and DBPs at 0, 1, 3 and 5 min of post intubation.

Laha *et al*. in their study compared dexmedetomidine 1 μg/kg with control and concluded that dexmedetomidine effectively blunted the hemodynamic responses during direct laryngoscopy and decreased the anaesthetic drug requisition.

From our study, we adequately establish that dexmedetomidine 1μg/kg was comparatively superior in attenuation of the haemodynamic changes during direct laryngoscopy.[55]

Now Dexmedetomidine has been started to use in the every section of anaesthesia because of its safety and effectiveness.

CONCLUSION

We conclude that dexmedetomidine in the dosage of 1 μg/kg over ten minutes before intubation efficiently attenuating the haemodynamic changes to laryngoscopy and endotracheal intubation.

Lignocaine in the dosage of 1.5 mg/kg given 3 min before laryngoscopy and intubation was not fully effective in reducing the increase in heart rate and blood pressure.

Dexmedetomidine 1 μg/kg has proved to keep the haemodynamics in stable manner during laryngoscopy & intubation. Hence Dexmedetomidine may beneficial for cardiac patients where the haemodynamic response to laryngoscopy and intubation is highly detrimental.

In brief dexmedetomidine is a highly selective α2 agonist has many desirable clinical benefits that encourage its use in the perioperative period.

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PROFORMA

Name: Age: years Sex: M/F

Weight: ASAPS: Date:

Surgery planned: IP. No:

Randomisation Code/ Envelope No :

Drug Used : D Group/ L Group

Mallampati score :

Time to Tracheal Intubation :

Neck manipulation :

Measured Outcomes :

	Prior Intubation		Post Intubation			
PARAMETER	Basal	PL*	0 min	1min	3min	5 min
Heart Rate						
Systolic Blood Pressure						
Diastolic Blood Pressure						
Mean Blood Pressure						

*PL – Pre Laryngoscopy

MASTER CHART

S NO	AGE	SEX	GROUP	WT	HT	BMI	BHR	BSBP	BDBP	BMBP	PLHR	PLSBP	PLDBP	PLMBP	0HR	0SBP	0DBP	0MBP	1HR	1SBP	1DBP	1MBP	3HR	3SBP	3DBP	3MBP	5HR	5SBP	5DBP	5MBP
1	58	F	L	78	172	26.4	103	140	88	105	101	130	81	97	105	126	74	91	97	120	77	91	96	116	74	88	94	112	63	79
2	32	M	L	71	169	24.9	104	138	84	102	102	129	79	96	105	115	72	86	98	110	69	83	96	105	65	78	94	102	63	76
3	60	M	L	54	160	21.1	99	134	80	98	97	126	74	91	102	130	73	92	92	124	74	91	89	118	74	89	86	118	59	79
4	18	M	L	69	170	23.9	83	112	71	85	81	104	64	77	85	109	65	80	84	103	62	76	82	98	60	73	80	98	54	69
5	22	M	L	78	176	25.2	73	104	63	77	71	98	63	75	74	108	63	78	72	102	60	74	74	98	61	73	74	99	54	69
6	40	M	L	54	156	22.2	63	102	61	75	61	94	65	75	63	109	62	78	64	101	62	75	67	94	58	70	66	92	57	69
7	46	F	L	76	175	24.8	70	98	62	74	68	100	59	73	72	106	62	77	70	99	56	70	68	90	58	69	68	90	50	63
8	33	M	L	64	157	26.0	75	100	61	74	73	91	60	70	76	98	56	70	71	93	54	67	74	91	55	67	77	90	49	63
9	55	F	L	68	168	24.1	72	104	62	76	69	96	61	73	71	105	63	77	70	99	55	70	72	90	53	65	75	89	50	63
10	32	M	L	76	174	25.1	67	109	69	82	65	98	63	75	69	109	70	83	69	100	63	75	72	96	54	68	73	92	50	64
11	42	M	L	69	176	22.3	91	120	73	89	89	109	66	80	92	117	72	87	84	109	66	80	83	102	55	71	80	98	55	69
12	30	M	L	58	162	22.1	82	110	66	81	80	102	60	74	85	109	69	82	82	100	62	75	80	92	54	67	77	88	53	65
13	28	M	L	58	162	22.1	93	126	79	95	91	118	70	86	95	121	74	90	88	111	67	82	85	104	62	76	82	90	56	67
14	37	M	L	75	179	23.4	76	102	62	75	74	95	60	72	77	109	63	78	83	101	62	75	88	96	55	69	86	90	58	69
15	52	M	L	60	172	20.3	78	105	63	77	76	97	56	70	81	108	66	80	74	99	53	68	71	95	52	66	70	90	50	63
16	43	F	L	62	165	22.8	99	130	82	98	96	119	70	86	100	121	70	87	90	112	69	83	86	104	61	75	82	102	57	72
17	36	M	L	66	169	23.1	86	116	71	86	84	108	64	79	87	110	66	81	95	106	62	77	92	101	63	76	90	99	61	74
18	46	M	L	58	158	23.2	80	107	64	78	77	94	60	71	82	103	62	76	76	98	54	69	73	94	52	66	70	92	48	63
19	40	M	L	66	172	22.3	71	101	64	76	69	92	59	70	73	103	60	74	72	99	53	68	75	95	55	68	77	96	50	65
20	45	M	L	64	164	23.8	79	106	65	79	76	99	61	74	79	107	62	77	81	103	59	74	77	99	57	71	74	100	51	67
21	60	F	L	64	164	23.8	84	113	68	83	82	105	60	75	87	105	62	76	80	100	57	71	77	97	54	68	75	96	51	66

MASTER CHART

S NO	AGE	SEX	GROUP	WT	HT	BMI	BHR	BSBP	BDBP	BMBP	PLHR	PLSBP	PLDBP	PLMBP	0HR	0SBP	0DBP	0MBP	1HR	1SBP	1DBP	1MBP	3HR	3SBP	3DBP	3MBP	5HR	5SBP	5DBP	5MBP
22	35	M	L	70	168	24.8	93	136	86	103	91	123	73	90	95	120	70	87	94	112	66	81	90	95	62	73	88	92	59	70
23	42	M	L	70	174	23.1	88	127	77	94	86	120	71	87	89	121	69	86	85	110	61	77	82	96	56	69	79	93	54	67
24	41	M	L	71	169	24.9	86	116	70	85	84	109	66	80	89	111	64	80	80	99	59	72	81	96	53	67	80	96	53	67
25	54	F	L	65	162	24.8	87	118	74	89	85	110	68	82	89	114	69	84	79	107	63	78	75	97	52	67	72	98	50	66
26	39	F	L	58	162	22.1	71	119	73	88	69	110	69	83	72	116	69	85	69	108	66	80	64	104	55	71	64	99	50	66
27	21	M	L	72	165	26.4	94	131	79	96	92	115	70	85	97	108	64	79	90	99	64	76	88	100	59	73	84	93	58	70
28	50	M	L	52	158	20.8	91	139	88	105	89	124	71	89	93	119	69	86	93	107	62	77	90	101	61	74	87	96	59	71
29	25	M	L	62	159	24.5	89	120	73	89	87	104	63	77	90	114	70	85	84	109	69	82	81	104	55	71	78	96	53	67
30	49	M	L	79	176	25.5	90	121	73	89	88	108	60	76	93	116	71	86	84	106	63	77	81	95	55	68	80	93	53	66
31	32	F	L	68	167	24.4	102	138	87	104	100	119	69	86	104	118	66	83	98	109	65	80	93	96	59	71	90	92	56	68
32	25	M	D	79	165	29.0	81	113	69	84	72	100	60	73	67	104	62	76	63	97	55	69	60	94	50	65	57	91	45	60
33	23	M	D	72	171	24.6	80	112	67	82	69	98	59	72	65	96	58	71	61	90	52	65	57	88	48	61	52	85	46	59
34	51	F	D	66	162	25.1	69	103	65	78	62	96	58	71	68	98	54	69	62	93	50	64	59	90	47	61	53	88	45	59
35	50	M	D	72	172	24.3	75	105	64	78	66	98	59	72	64	97	58	71	62	93	52	66	57	90	48	62	54	89	44	59
36	38	M	D	69	168	24.4	87	107	64	78	72	99	59	72	66	94	56	69	61	90	50	63	55	88	47	61	51	85	44	58
37	19	M	D	67	168	23.7	85	105	65	78	73	94	54	67	66	98	56	70	61	94	51	65	54	90	46	61	53	88	44	59
38	23	M	D	74	176	23.9	76	110	67	81	67	100	60	73	66	104	61	75	63	94	55	68	59	90	50	63	55	88	47	61
39	23	M	D	70	169	24.5	90	126	76	93	78	100	60	73	70	103	62	76	65	98	54	69	60	95	50	65	55	92	47	62
40	34	F	D	76	178	24.0	100	140	88	105	82	108	65	79	77	106	64	78	70	100	60	73	66	94	50	65	60	91	46	61

MASTER CHART

S NO	AGE	SEX	GROUP	WT	HT	BMI	BHR	BSBP	BDBP	BMBP	PLHR	PLSBP	PLDBP	PLMBP	0HR	0SBP	0DBP	0MBP	1HR	1SBP	1DBP	1MBP	3HR	3SBP	3DBP	3MBP	5HR	5SBP	5DBP	5MBP
41	21	M	D	69	174	22.8	79	110	67	81	70	98	59	72	67	100	60	73	62	96	54	68	59	92	50	64	55	91	48	62
42	52	M	D	69	172	23.3	79	103	62	76	64	94	56	69	62	96	58	71	58	96	52	67	55	92	47	62	51	90	45	60
43	32	M	D	70	171	23.9	78	109	69	82	71	102	61	75	65	96	58	71	61	98	53	68	57	92	48	63	54	90	45	60
44	40	F	D	69	170	23.9	96	130	79	96	82	104	60	75	77	106	61	76	70	99	53	68	63	95	50	65	57	90	46	61
45	33	M	D	80	177	25.5	102	141	85	104	90	104	62	76	82	107	64	78	74	100	54	69	67	97	49	65	60	94	44	61
46	23	M	D	62	168	22.0	81	130	82	98	74	110	66	81	68	108	65	79	63	102	60	74	61	99	53	68	55	97	48	64
47	20	M	D	64	163	24.1	95	133	81	98	86	110	66	81	81	102	61	75	77	98	53	68	72	94	51	65	64	91	48	62
48	25	M	D	72	168	25.5	97	135	81	99	87	112	67	82	83	107	64	78	76	103	58	73	70	98	50	66	63	92	46	61
49	32	F	D	68	164	25.3	90	126	79	95	83	107	64	78	77	102	61	75	73	98	54	69	66	95	49	64	60	92	45	61
50	47	M	D	65	169	22.8	83	116	71	86	74	102	61	75	71	104	62	76	66	100	55	70	63	97	50	66	59	94	46	62
51	33	M	D	63	157	25.6	72	120	78	92	62	96	54	68	58	100	61	74	54	94	53	67	60	90	50	63	50	87	47	60
52	45	M	D	67	178	21.1	77	107	67	80	70	98	59	72	66	96	58	71	62	90	52	65	60	88	47	61	55	86	45	59
53	52	F	D	70	168	24.8	73	110	62	78	67	97	55	69	62	98	58	71	59	93	52	66	56	90	50	63	53	85	48	60
54	41	F	D	69	174	22.8	78	109	65	80	68	100	60	73	64	101	63	76	60	94	53	67	58	92	48	63	54	90	46	61
55	52	M	D	69	165	25.3	86	120	76	91	79	100	60	73	78	102	61	75	72	94	57	69	67	90	51	64	63	86	47	60
56	29	M	D	64	164	23.8	88	123	75	91	79	101	61	74	74	98	62	74	67	97	55	69	63	88	50	63	59	84	48	60
57	23	M	D	71	168	25.2	91	127	76	93	81	102	61	75	77	100	60	73	71	97	59	72	66	92	47	62	62	88	44	59
58	40	M	D	66	164	24.5	95	124	78	93	88	113	68	83	82	108	65	79	75	102	60	74	69	98	50	66	60	96	46	63
59	35	M	D	64	159	25.3	93	120	73	89	84	108	65	79	79	102	61	75	73	98	55	69	69	93	48	63	63	90	46	61
60	19	M	D	62	155	25.8	101	130	78	95	85	102	61	75	81	100	60	73	70	94	57	69	64	90	46	61	56	89	42	58
61	40	M	D	69	166	25.0	99	123	77	92	88	105	66	79	82	102	61	75	72	94	56	69	70	90	48	62	61	88	46	60